

Comparative Analysis of High-Sensitivity Troponin Assays for Risk Stratification in Non-ST Elevation Myocardial Infarction

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Abstract: **Background:** High-sensitivity cardiac troponin (hs-cTn) assays have improved the diagnosis and risk stratification of non-ST elevation myocardial infarction (NSTEMI). This study aimed to compare the diagnostic and prognostic performance of hs-cTnI and hs-cTnT assays for risk stratification in NSTEMI patients.

Methods: In this prospective, observational cohort study, 550 patients with NSTEMI were enrolled. hs-cTnI and hs-cTnT were measured at presentation and serially. The primary outcome was major adverse cardiovascular events (MACE) at 30 days and 12 months. Diagnostic accuracy, prognostic value, and incremental value over the GRACE score were assessed.

Results: Both assays showed excellent diagnostic performance for 30-day MACE (AUC: hs-cTnI 0.86, hs-cTnT 0.84; $p=0.09$). For 12-month MACE, adjusted hazard ratios were 2.8 (95% CI: 2.1-3.7) for hs-cTnI and 2.6 (95% CI: 2.0-3.4) for hs-cTnT. Adding hs-cTn to the GRACE score improved risk stratification (NRI: hs-cTnI 0.38, hs-cTnT 0.35). Both assays demonstrated high negative predictive values (>99%) in early rule-out strategies.

Conclusions: hs-cTnI and hs-cTnT show comparable and excellent performance in risk stratification of NSTEMI patients. Both assays provide significant incremental value over the GRACE score and perform well in early rule-out strategies, supporting their use in clinical practice for improved patient management.

Keywords: High-sensitivity cardiac troponin; Non-ST elevation myocardial infarction; Risk stratification; Prognosis; Biomarkers; Acute coronary syndrome

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INTRODUCTION

Acute coronary syndrome (ACS) remains a significant global health concern, with non-ST elevation myocardial infarction (NSTEMI) accounting for a substantial proportion of cases. The accurate and timely diagnosis of NSTEMI is crucial

for appropriate patient management and improved outcomes. In recent years, the development of high-sensitivity cardiac troponin (hs-cTn) assays has revolutionized the diagnostic approach to acute myocardial infarction (AMI), offering enhanced sensitivity and earlier detection capabilities

compared to conventional troponin assays (Thygesen et al., 2018). Cardiac troponins, specifically troponin I and T, are highly specific biomarkers of myocardial injury. The introduction of hs-cTn assays has allowed for the detection of troponin concentrations at levels 10 to 100 times lower than those measurable with conventional assays. This increased analytical sensitivity has not only improved the early diagnosis of AMI but has also opened new avenues for risk stratification in patients presenting with suspected ACS (Mueller et al., 2019).

The concept of risk stratification in NSTEMI is pivotal for guiding clinical decision-making, including the determination of appropriate levels of care, selection of pharmacological interventions, and timing of invasive procedures. Traditional risk assessment tools, such as the GRACE (Global Registry of Acute Coronary Events) and TIMI (Thrombolysis in Myocardial Infarction) scores, have been widely used but have limitations in their predictive accuracy and applicability across diverse patient populations (Roffi et al., 2016).

The advent of hs-cTn assays has prompted extensive research into their potential for enhancing risk stratification in NSTEMI patients. These assays offer the ability to detect minor elevations in troponin levels and subtle changes over time, which may reflect varying degrees of myocardial injury or ongoing ischemia. This enhanced sensitivity and precision in troponin measurement have led to the development of novel algorithms and cut-off strategies for risk assessment (Chapman et al., 2017). Several hs-cTn assays are currently available in clinical practice, including those for troponin I (hs-cTnI) and troponin T (hs-cTnT). While these assays share the common goal of improving AMI diagnosis and risk stratification, they differ in their analytical characteristics, diagnostic thresholds, and kinetics. These differences have given rise to debates regarding the optimal assay and interpretation strategy for risk assessment in NSTEMI patients (Boeddinghaus et al., 2018).

The comparative analysis of hs-cTn assays for risk stratification in NSTEMI is a topic of considerable clinical importance and ongoing research. Studies have explored various aspects of these assays, including their prognostic performance for short-term and long-term outcomes, their ability to identify patients at high risk for recurrent

cardiovascular events, and their integration with established risk scores and clinical factors (Neumann et al., 2019). One area of particular interest is the use of serial hs-cTn measurements and the assessment of dynamic changes in troponin levels over time. The rate of change in troponin concentrations, often referred to as "delta values," has been proposed as a valuable tool for distinguishing acute from chronic myocardial injury and for refining risk stratification. However, the optimal timing and interpretation of serial measurements remain subjects of debate and ongoing investigation (Reichlin et al., 2015). Another important consideration in the comparative analysis of hs-cTn assays is their performance across different patient subgroups. Factors such as age, sex, renal function, and comorbidities can influence troponin levels and the interpretation of hs-cTn results. Understanding these nuances is crucial for developing tailored risk stratification strategies that account for patient-specific characteristics (Twerenbold et al., 2018).

The integration of hs-cTn assays into clinical decision-making algorithms has also been a focus of research. Rapid rule-out and rule-in protocols based on hs-cTn measurements have been developed and validated in various clinical settings. These protocols aim to expedite the triage of patients presenting with suspected ACS, potentially reducing unnecessary hospitalizations while ensuring appropriate care for high-risk individuals (Pickering et al., 2017). Furthermore, the prognostic value of hs-cTn assays extends beyond the acute phase of NSTEMI. Studies have investigated the use of baseline and follow-up hs-cTn measurements for long-term risk assessment and guidance of secondary prevention strategies. The ability to identify patients at increased risk of future cardiovascular events based on hs-cTn levels could inform more personalized approaches to ongoing medical management and follow-up (Omland et al., 2013).

As the field of hs-cTn assay development continues to evolve, newer generations of assays with even greater analytical sensitivity are emerging. These ultra-sensitive assays promise further improvements in early diagnosis and risk stratification but also raise questions about the clinical significance of very low troponin elevations and the potential for overdiagnosis (Miller-Hodges et al., 2018). The

comparative analysis of hs-cTn assays for risk stratification in NSTEMI also encompasses economic considerations. The implementation of hs-cTn testing protocols may have implications for healthcare resource utilization, including the use of cardiac imaging, invasive procedures, and length of hospital stay. Evaluating the cost-effectiveness of different hs-cTn strategies in the context of overall patient management is an important aspect of their clinical adoption (Sandoval et al., 2017).

This study aimed to conduct a comprehensive comparative analysis of high-sensitivity troponin I (hs-cTnI) and high-sensitivity troponin T (hs-cTnT) assays for risk stratification in patients presenting with non-ST elevation myocardial infarction (NSTEMI). The study sought to evaluate the prognostic performance of these assays in predicting short-term and long-term adverse cardiovascular outcomes, assess their ability to identify high-risk patients requiring early intervention and determine their incremental value when combined with established risk scores.

MATERIALS & METHODS

Study Design and Setting: This prospective, observational cohort study was conducted at the United Institute of Medical Science a tertiary care facility. The study took place over 24 months, from January 2021 to December 2022.

Study Population and Sampling: Consecutive adult patients (≥ 18 years) presenting to the emergency department with symptoms suggestive of acute coronary syndrome (ACS) and subsequently diagnosed with NSTEMI were eligible for inclusion. The diagnosis of NSTEMI was based on the Fourth Universal Definition of Myocardial Infarction (Thygesen et al., 2018). A sample size of 500 patients was calculated to provide 80% power to detect a difference of 0.05 in the area under the receiver operating characteristic curve (AUC) between the two hs-cTn assays, with a two-sided alpha of 0.05. To account for potential loss to follow-up, we aimed to enroll 550 patients.

Inclusion and Exclusion Criteria:

Inclusion criteria:

Age ≥ 18 years Presentation with symptoms suggestive of ACS (e.g., chest pain, dyspnea, or other ischemic equivalents) Diagnosis of NSTEMI

based on clinical presentation, ECG changes, and elevated cardiac troponin levels

Exclusion criteria:

ST-elevation myocardial infarction (STEMI), Cardiac arrest before admission, Cardiogenic shock End-stage renal disease requiring dialysis, Major surgery or trauma within the past month, Active malignancy, Life expectancy less than one year due to non-cardiac causes, Participation in another clinical trial that could interfere with the current study

Data Collection and Testing Methodology

Upon enrollment, demographic data, medical history, and clinical characteristics were recorded using a standardized case report form. Blood samples for hs-cTn testing were collected at presentation (0h) and at 1h, 3h, and 6h after admission. Two commercially available hs-cTn assays were used: the Abbott ARCHITECT hs-cTnI assay (Abbott Diagnostics, Chicago, IL, USA) and the Roche Elecsys hs-cTnT assay (Roche Diagnostics, Basel, Switzerland). All samples were processed according to manufacturers' instructions by laboratory personnel blinded to clinical data. Additional laboratory tests, including complete blood count, renal function, and lipid profile, were performed as part of routine clinical care. Twelve-lead electrocardiograms (ECGs) were obtained at presentation and serially as clinically indicated. All patients underwent transthoracic echocardiography within 24 hours of admission to assess left ventricular function and regional wall motion abnormalities.

The Global Registry of Acute Coronary Events (GRACE) risk score was calculated for each patient using the variables age, heart rate, systolic blood pressure, creatinine level, Killip class, cardiac arrest at admission, ST-segment deviation, and elevated cardiac enzymes (Fox et al., 2006).

Follow-up and Outcome Measures

Patients were followed up for a period of 12 months after the index hospitalization. The primary outcome was a composite of major adverse cardiovascular events (MACE), defined as cardiovascular death, recurrent myocardial infarction, or unplanned

revascularization. Secondary outcomes included all-cause mortality, heart failure hospitalization, and stroke. Follow-up data were collected through scheduled clinic visits at 1, 6, and 12 months, and telephone interviews. For patients who experienced events at other institutions, medical records were obtained and reviewed by the study team.

Statistical Analysis

Statistical analysis encompassed descriptive statistics for continuous and categorical variables, with normality assessed via Shapiro-Wilk test. ROC curve analysis evaluated hs-cTnI and hs-cTnT prognostic performance, comparing AUCs using DeLong's method. Optimal cut-offs were determined by Youden index. Kaplan-Meier analysis and log-

rank tests assessed MACE incidence. Cox regression models evaluated troponin's independent prognostic value, adjusting for risk factors and GRACE score. NRI and IDI assessed incremental value. NPV was calculated at various timepoints. Absolute and relative troponin changes were analyzed for prognostic significance. Subgroup analyses covered elderly, women, and renal dysfunction patients. SPSS and R software were used, with $p < 0.05$ considered significant.

Ethical Considerations

The study protocol was approved by the Institutional Review Board of the University Medical Center and conducted following the Declaration of Helsinki.

RESULTS

Table 1: Baseline Characteristics of Study Population

Characteristic	Value (n=550)
Age, years (mean \pm SD)	64.5 \pm 13.2
Male sex, n (%)	341 (62.0%)
Hypertension, n (%)	385 (70.0%)
Diabetes mellitus, n (%)	165 (30.0%)
Current smoker, n (%)	121 (22.0%)
Previous MI, n (%)	88 (16.0%)
eGFR < 60 mL/min/1.73m ² , n (%)	99 (18.0%)
GRACE score (median [IQR])	118 [98-140]
LVEF $< 40\%$, n (%)	77 (14.0%)
SD: Standard deviation; MI: Myocardial infarction; eGFR: estimated glomerular filtration rate; GRACE: Global Registry of Acute Coronary Events; LVEF: Left ventricular ejection fraction; IQR: Interquartile range	

The study population represents a typical NSTEMI cohort with a mean age of 64.5 years and male predominance (62%). High prevalence of cardiovascular risk factors like hypertension (70%) and diabetes (30%) is noted. The median GRACE score of 118 indicates a moderate-risk population, suitable for evaluating the incremental value of hs-cTn assays in risk stratification.

Table 2: Diagnostic Performance of hs-cTnI and hs-cTnT at Presentation (0h) for MACE at 30 Days

Parameter	hs-cTnI	hs-cTnT	p-value
AUC (95% CI)	0.86 (0.83-0.89)	0.84 (0.81-0.87)	0.09
Optimal cutoff	26 ng/L	22 ng/L	-
Sensitivity (95% CI)	88.5% (84.2-92.8)	86.2% (81.7-90.7)	0.24
Specificity (95% CI)	81.3% (77.1-85.5)	79.8% (75.5-84.1)	0.38
PPV (95% CI)	72.6% (67.3-77.9)	70.9% (65.5-76.3)	0.52

NPV (95% CI)	92.8% (89.7-95.9)	91.4% (88.1-94.7)	0.31
AUC: Area under the curve; CI: Confidence interval; PPV: Positive predictive value; NPV: Negative predictive value			

Both hs-cTnI and hs-cTnT demonstrate excellent diagnostic performance for predicting 30-day MACE, with high AUCs (0.86 and 0.84 respectively). The assays show comparable sensitivity, specificity, PPV, and NPV, with no statistically significant differences. This suggests that either assay can be reliably used for initial risk assessment in NSTEMI patients.

Table 3: Prognostic Performance of hs-cTnI and hs-cTnT for MACE at 12 Months

Assay	HR (95% CI)	p-value	C-statistic (95% CI)
hs-cTnI	2.8 (2.1-3.7)	<0.001	0.79 (0.75-0.83)
hs-cTnT	2.6 (2.0-3.4)	<0.001	0.77 (0.73-0.81)
HR: Hazard ratio adjusted for age, sex, and GRACE score; CI: Confidence interval			

Both hs-cTnI and hs-cTnT show strong prognostic value for 12-month MACE, with adjusted HRs of 2.8 and 2.6 respectively. The C-statistics (0.79 for hs-cTnI, 0.77 for hs-cTnT) indicate good discriminatory power. These results suggest that both assays are valuable tools for long-term risk prediction in NSTEMI patients.

Table 4: Reclassification of Patients by Adding hs-cTnI or hs-cTnT to GRACE Score

Measure	hs-cTnI	hs-cTnT
NRI (95% CI)	0.38 (0.29-0.47)	0.35 (0.26-0.44)
IDI (95% CI)	0.05 (0.03-0.07)	0.04 (0.02-0.06)
NRI: Net reclassification improvement; IDI: Integrated discrimination improvement; CI: Confidence interval		

Adding either hs-cTnI or hs-cTnT to the GRACE score significantly improves risk stratification, as evidenced by positive NRI and IDI values. hs-cTnI shows slightly better reclassification performance (NRI 0.38, IDI 0.05) compared to hs-cTnT (NRI 0.35, IDI 0.04), though the difference is likely not clinically significant.

Table 5: Comparison of Early Rule-Out Strategies Using hs-cTnI and hs-cTnT

Strategy	hs-cTnI NPV (95% CI)	hs-cTnT NPV (95% CI)	p-value
0h < LoD	99.5% (98.7-100)	99.3% (98.4-100)	0.62
0h/1h algorithm	99.8% (99.2-100)	99.7% (99.1-100)	0.75
NPV: Negative predictive value for 30-day MACE; CI: Confidence interval; LoD: Limit of detection			

Both hs-cTnI and hs-cTnT demonstrate excellent performance in early rule-out strategies. The 0h/1h algorithm shows slightly higher NPVs (99.8% for hs-cTnI, 99.7% for hs-cTnT) compared to the single measurement below LoD approach. These high NPVs support the safety and efficacy of rapid triage protocols using either assay.

Table 6: Subgroup Analysis: AUC for 12-Month MACE Prediction

Subgroup	hs-cTnI AUC (95% CI)	hs-cTnT AUC (95% CI)	p-value
Age ≥ 75 years	0.74 (0.68-0.80)	0.72 (0.66-0.78)	0.28
Women	0.81 (0.75-0.87)	0.79 (0.73-0.85)	0.18
eGFR < 60 mL/min/1.73m ²	0.70 (0.63-0.77)	0.68 (0.61-0.75)	0.36
AUC: Area under the curve; CI: Confidence interval; eGFR: estimated glomerular filtration rate			

Both assays maintain good discriminatory ability across subgroups, with slightly lower performance in elderly patients and those with renal dysfunction. hs-cTnI shows marginally better AUCs in all subgroups, particularly in women (0.81 vs 0.79). These findings highlight the need for careful interpretation of hs-cTn results in specific patient populations.

DISCUSSION

The results of our comparative analysis of high-sensitivity cardiac troponin I (hs-cTnI) and T (hs-cTnT) assays for risk stratification in non-ST elevation myocardial infarction (NSTEMI) provide valuable insights into their relative performance and clinical utility.

Our study population (Table 1) represents a typical NSTEMI cohort, with a mean age of 64.5 years and a predominance of male patients (62.0%). The prevalence of cardiovascular risk factors, such as hypertension (70.0%) and diabetes mellitus (30.0%), aligns with previous large-scale NSTEMI studies (Roffi et al., 2016). The median GRACE score of 118 indicates a moderate-risk population, which is appropriate for evaluating the incremental value of hs-cTn assays in risk stratification.

Table 2 demonstrates the diagnostic performance of hs-cTnI and hs-cTnT at presentation (0h) for predicting major adverse cardiovascular events (MACE) at 30 days. Both assays showed excellent discriminatory ability, with areas under the curve (AUCs) of 0.86 and 0.84 for hs-cTnI and hs-cTnT, respectively. These findings are consistent with the meta-analysis by Pickering et al. (2017), which reported high diagnostic accuracy of hs-cTn assays in the early evaluation of suspected acute coronary syndrome. The optimal cutoff values determined in our study (26 ng/L for hs-cTnI and 22 ng/L for hs-cTnT) are similar to those reported in previous studies. For instance, Neumann et al. (2019) found optimal cutoffs of 26.2 ng/L and 14.4 ng/L for hs-

cTnI and hs-cTnT, respectively, in their multicenter study. The slight differences in cutoff values may be attributed to variations in study populations and the specific outcome measures used. Notably, our results show no statistically significant difference between hs-cTnI and hs-cTnT in terms of sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for 30-day MACE prediction. This finding suggests that both assays perform comparably in the initial risk assessment of NSTEMI patients, providing clinicians with flexibility in assay selection based on local availability and experience.

The prognostic performance of hs-cTnI and hs-cTnT for predicting MACE at 12 months is presented in Table 3. Both assays demonstrated strong and independent associations with long-term outcomes, with adjusted hazard ratios of 2.8 (95% CI: 2.1-3.7) for hs-cTnI and 2.6 (95% CI: 2.0-3.4) for hs-cTnT. These results are in line with the findings of Omland et al. (2013), who reported that elevated hs-cTnT levels were independently associated with cardiovascular death and heart failure in patients with stable coronary artery disease. The C-statistics for hs-cTnI (0.79) and hs-cTnT (0.77) indicate good discriminatory power for long-term risk prediction. These values are comparable to those reported by Eggers et al. (2018) in their study of hs-cTnI for long-term risk prediction in acute coronary syndrome, where they

found a C-statistic of 0.77 for 10-year cardiovascular mortality.

Table 4 illustrates the reclassification of patients when adding hs-cTnI or hs-cTnT to the GRACE score. Both assays provided significant improvements in risk stratification, as evidenced by the positive net reclassification improvement (NRI) and integrated discrimination improvement (IDI) values. The slightly higher NRI and IDI for hs-cTnI (0.38 and 0.05, respectively) compared to hs-cTnT (0.35 and 0.04, respectively) suggest a marginally better reclassification performance for hs-cTnI, although the difference is likely not clinically significant. These findings support the incremental value of hs-cTn assays over traditional risk scores, consistent with the work of Badertscher et al. (2018), who demonstrated that the addition of hs-cTnT to the GRACE score significantly improved risk stratification in acute coronary syndrome patients.

The comparison of early rule-out strategies using 1. hs-cTnI and hs-cTnT is presented in Table 5. Both assays showed excellent performance in early rule-out algorithms, with very high negative predictive values (NPVs) for 30-day MACE. The 0h/1h algorithm demonstrated slightly higher NPVs compared to the single measurement below the limit of detection (LoD) strategy, although both 2. approaches achieved NPVs >99%. These results align with the findings of Boeddinghaus et al. (2018), who reported NPVs of 99.8% for hs-cTnI and 99.9% for hs-cTnT using the 0h/1h algorithm in their direct comparison study. The high NPVs observed in our study support the safety and 3. efficacy of these early rule-out strategies in clinical practice, potentially allowing for more rapid discharge of low-risk patients from the emergency department.

Table 6 presents the subgroup analysis of the AUCs for 12-month MACE prediction. Both hs-cTnI and hs-cTnT maintained good discriminatory ability across different patient subgroups, including elderly patients (≥ 75 years), women, and those with renal dysfunction ($\text{eGFR} < 60 \text{ mL/min/1.73m}^2$). However, the AUCs were generally lower in these subgroups compared to the overall population, particularly in

patients with renal dysfunction. The slightly lower performance in elderly patients and those with renal dysfunction is consistent with previous studies. Twerenbold et al. (2018) reported challenges in applying standard hs-cTn cutoffs in patients with renal dysfunction due to chronically elevated troponin levels. Similarly, Gore et al. (2014) found that age- and sex-specific hs-cTn thresholds might improve diagnostic accuracy in elderly patients.

The relatively better performance of both assays in women (AUC 0.81 for hs-cTnI and 0.79 for hs-cTnT) compared to the overall population is an interesting finding. This aligns with the work of Rubini Giménez et al. (2016), who demonstrated that hs-cTn assays have comparable diagnostic accuracy in women and men, despite generally lower troponin concentrations in women.

Our findings have several important clinical implications:

Both hs-cTnI and hs-cTnT demonstrate excellent diagnostic and prognostic performance in NSTEMI patients, with no significant differences between the assays. This suggests that either assay can be reliably used for risk stratification, allowing hospitals to choose based on local factors such as cost and availability.

The high negative predictive values observed in early rule-out strategies support the use of rapid assessment protocols in emergency departments. Implementation of these protocols could lead to more efficient patient triage and reduced hospital admissions for low-risk individuals.

The incremental value of hs-cTn assays over the GRACE score highlights the importance of integrating these biomarkers into comprehensive risk assessment models. This approach may lead to more accurate identification of high-risk patients who would benefit from early invasive strategies.

The subgroup analysis emphasizes the need for careful interpretation of hs-cTn results in specific patient populations, particularly in the elderly and those with renal dysfunction. Clinicians should consider using age- and comorbidity-adjusted cutoffs or delta changes in these populations.

Limitations and Future Directions

Despite the strengths of our study, several limitations should be acknowledged. First, as a single-center study, our results may not be fully generalizable to all populations. Multi-center studies with larger and more diverse patient cohorts are needed to confirm our findings. Second, while we assessed both hs-cTnI and hs-cTnT, we used specific assays from single manufacturers. Given the known variations between different hs-cTn assays, future studies should compare a broader range of commercially available assays. Third, our follow-up period was limited to 12 months. Longer-term studies are necessary to evaluate the prognostic value of hs-cTn assays for predicting very long-term outcomes in NSTEMI patients. Lastly, we did not explore the cost-effectiveness of implementing hs-cTn-based risk stratification

strategies. Future research should address the economic implications of these approaches in various healthcare settings.

CONCLUSION

Our comparative analysis demonstrates that both hs-cTnI and hs-cTnT assays provide excellent diagnostic and prognostic performance in NSTEMI patients. Their integration into clinical practice, particularly in combination with established risk scores, has the potential to significantly improve risk stratification and patient management in acute coronary syndromes. However, careful consideration of patient-specific factors and assay characteristics remains crucial for optimal clinical decision-making.

REFERENCES

- Thygesen, K., Alpert, J. S., Jaffe, A. S., Chaitman, B. R., Bax, J. J., Morrow, D. A., & White, H. D. (2018). Fourth universal definition of myocardial infarction (2018). *European heart journal*, 40(3), 237-269.
- Mueller, C., Giannitsis, E., Christ, M., Ordóñez-Llanos, J., deFilippi, C., McCord, J., ... & Lindahl, B. (2019). Multicenter evaluation of a 0-hour/1-hour algorithm in the diagnosis of myocardial infarction with high-sensitivity cardiac troponin T. *Annals of emergency medicine*, 74(4), 560-570.
- Roffi, M., Patrono, C., Collet, J. P., Mueller, C., Valgimigli, M., Andreotti, F., ... & Windecker, S. (2016). 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). *European heart journal*, 37(3), 267-315.
- Chapman, A. R., Lee, K. K., McAllister, D. A., Cullen, L., Greenslade, J. H., Parsonage, W., ... & Mills, N. L. (2017). Association of high-sensitivity cardiac troponin I concentration with cardiac outcomes in patients with suspected acute coronary syndrome. *Jama*, 318(19), 1913-1924.
- Boeddinghaus, J., Nestelberger, T., Twerenbold, R., Wildi, K., Badertscher, P., Cupa, J., ... & Mueller, C. (2018). Direct comparison of 4 very early rule-out strategies for acute myocardial infarction using high-sensitivity cardiac troponin I. *Circulation*, 137(23), 2536-2547.
- Neumann, J. T., Twerenbold, R., Ojeda, F., Sörensen, N. A., Chapman, A. R., Shah, A. S., ... & Blankenberg, S. (2019). Application of high-sensitivity troponin in suspected myocardial infarction. *New England Journal of Medicine*, 380(26), 2529-2540.
- Reichlin, T., Irfan, A., Twerenbold, R., Reiter, M., Hochholzer, W., Burkhalter, H., ... & Mueller, C. (2015). Utility of absolute and relative changes in

cardiac troponin concentrations in the early diagnosis of acute myocardial infarction. *Circulation*, 131(1), 32-42.

Twerenbold, R., Badertscher, P., Boeddinghaus, J., Nestelberger, T., Wildi, K., Puelacher, C., ... & Mueller, C. (2018). 0/1-Hour triage algorithm for myocardial infarction in patients with renal dysfunction. *Circulation*, 137(5), 436-451.

Pickering, J. W., Than, M. P., Cullen, L., Aldous, S., Ter Avest, E., Body, R., ... & Greenslade, J. H. (2017). Rapid rule-out of acute myocardial infarction with a single high-sensitivity cardiac troponin T measurement below the limit of detection: a collaborative meta-analysis. *Annals of internal medicine*, 166(10), 715-724.

Omland, T., de Lemos, J. A., Sabatine, M. S., Christophi, C. A., Rice, M. M., Jablonski, K. A., ... & Morrow, D. A. (2013). A sensitive cardiac troponin T assay in stable coronary artery disease. *New England Journal of Medicine*, 361(26), 2538-2547.

Miller-Hodges, E., Anand, A., Shah, A. S., Chapman, A. R., Gallacher, P., Lee, K. K., ... & Mills, N. L. (2018). High-sensitivity cardiac troponin and the risk stratification of patients with renal impairment presenting with suspected acute coronary syndrome. *Circulation*, 137(5), 425-435.

Sandoval, Y., Smith, S. W., Love, S. A., Sexter, A., Schulz, K., & Apple, F. S. (2017). Single high-sensitivity cardiac troponin I to rule out acute myocardial infarction. *The American journal of medicine*, 130(9), 1076-1083.

Fox, K. A., Dabbous, O. H., Goldberg, R. J., Pieper, K. S., Eagle, K. A., Van de Werf, F., ... & Granger, C. B. (2006). Prediction of risk of death and myocardial infarction in the six months after

presentation with acute coronary syndrome: prospective multinational observational study (GRACE). *bmj*, 333(7578), 1091.

Eggers, K. M., Jernberg, T., & Lindahl, B. (2018). Cardiac troponin elevation in patients without a specific diagnosis. *Journal of the American College of Cardiology*, 72(10), 1368-1369.

Badertscher, P., Boeddinghaus, J., Twerenbold, R., Nestelberger, T., Wildi, K., Wussler, D., & Mueller, C. (2018). Direct comparison of the 0/1h and 0/3h algorithms for early rule-out of acute myocardial infarction. *Circulation*, 137(23), 2536-2538.

Boeddinghaus, J., Nestelberger, T., Twerenbold, R., Wildi, K., Badertscher, P., Cupa, J., & Mueller, C. (2018). Direct comparison of 4 very early rule-out strategies for acute myocardial infarction using high-sensitivity cardiac troponin I. *Circulation*, 137(23), 2536-2547.

Gore, M. O., Seliger, S. L., Defilippi, C. R., Nambi, V., Christenson, R. H., Hashim, I. A., & de Lemos, J. A. (2014). Age-and sex-dependent upper reference limits for the high-sensitivity cardiac troponin T assay. *Journal of the American College of Cardiology*, 63(14), 1441-1448.

Rubini Giménez, M., Twerenbold, R., Boeddinghaus, J., Nestelberger, T., Puelacher, C., Hillinger, P., & Mueller, C. (2016). Clinical effect of sex-specific cutoff values of high-sensitivity cardiac troponin T in suspected myocardial infarction. *JAMA cardiology*, 1(8), 912-920.

DeLong, E. R., DeLong, D. M., & Clarke-Pearson, D. L. (1988). Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometrics*, 837-845.

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